Anomalous High Origin of the Right Coronary Artery Above the Sinotubular Junction

Veysel Tosun,1 Necmettin Korucuk,2 Unal Guntekin3

1Department of Cardiology, Sanliurfa Training and Research Hospital, Sanliurfa, Turkey  
2Department of Cardiology, Ercis State Hospital, Van, Turkey  
3Department of Cardiology, Akdeniz University Faculty of Medicine, Antalya, Turkey

Abstract

Abnormal origin of the right coronary artery (RCA) from the left side of ascending aorta and continuing between the aorta and the truncus pulmonalis is a very rare congenital anomaly. Systolic expansion of the aorta and pulmonary trunk may lead to compression of the coronary artery and result in myocardial ischemia, particularly with exertion. A 59-year-old man admitted to cardiology department with prolonged chest pain during exercise. Abnormal origin of RCA with an interarterial course between the aorta and pulmonary artery was observed on coronary angiography and multi-detector computed tomography coronary angiography (MCTCA). In addition, RCA output compression was reported on MCTCA.

Keywords: Anomalous origin of coronary artery, multidetector computed tomography coronary angiography, myocardial ischaemia

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Case Report

A 59-year-old man was admitted to our clinic with prolonged chest pain particularly during exercise. The patient’s history included surgery for large cell lung cancer in 1994 and a dual-chamber (DDD) pacemaker implantation for a complete atrioventricular block in 2010. His physical examination was normal, and myocardial enzyme levels were not elevated. Electrocardiography showed paced rhythm (atrial sense, ventricular paced, 90 beats/min). There were no any pathological findings on transthoracic echocardiography, except mild valve insufficiencies. Coronary angiography by the femoral approach demonstrated non-critical plaques in LMCA, LAD, and Cx arteries. RCA could not be viewed using standard JR catheters. After ascending aortography with a 6-Fr pigtail catheter, RCA could be viewed with 6-Fr Amplatz Left 2 guiding catheter. RCA originated from the left side of ascending aorta above the sinotubular junction (not from the sinus of Valsalva) (Fig. 1 and 2). There was a 40% stenosis of the proximal RCA. He underwent multi-detector computed tomography coronary angiography (MCTCA) to exclude obstructive coronary artery

Address for correspondence: Veysel Tosun, MD. Sanliurfa Egitim ve Arastirma Hastanesi Kardiyoloji Bolumu, Sanliurfa, Turkey  
Phone: +90 414 317 17 17 E-mail: veysetosun8810@gmail.com

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disease. MCTCA revealed 25% stenosis of the left main coronary artery, up to 70% stenosis of the left coronary arteries, and abnormal origin of RCA with an interarterial course between the aorta and pulmonary artery (Fig. 3). In addition, RCA output compression was reported on MCTCA. We did not perform a stress test to investigate the effect of the interarterial course of RCA because the patient has pacemaker. Antiplatelet and antianginal therapy were initiated and the patient was discharged.

**Discussion**

A study that reviewed seven different studies showed that the incidence of anomalous RCA in patients undergoing coronary angiography was between 0.07% and 0.46%. Most of the anomalies were RCA originating from the left sinus of Valsalva. Fatal or nonfatal myocardial infarction and sudden death occur in up to 30% cases of anomalous origin of RCA in the absence of coronary atherosclerosis. In vivo identification of anomalous coronary artery disease is still a challenge because of insufficient warning signs. Computed tomographic angiography with three-dimensional reconstruction is helpful for demonstrating the spatial relationship of anomalous arteries to the aortic root. Cardiac magnetic resonance imaging might be another helpful modality.

Manifestations vary from asymptomatic patients to those who present with angina pectoris, myocardial infarction, heart failure, syncope, arrhythmias, and also sudden death. Myocardial ischemia in association with this anomaly is thought to be caused by an abnormal RCA and angulations and compression of RCA between the aorta and pulmonary trunk during exercise.

Our patient demonstrated the interarterial type anomaly. RCA originated from the left side of ascending aorta (not from the sinus of Valsalva) and continued between the aorta and truncus pulmonalis. An association is often found between this type of abnormal course of RCA and increasing myocardial ischemia, particularly during severe
exertion. Outward expansion of the roots of the aorta and pulmonary trunk can lead to compression of the coronary arteries. Abnormal mechanical stresses and flow patterns may result in internal injury, producing atherosclerotic changes in the anomalous segments. This altered myocardial oxygen supply-demand in such anomalous coronaries can worsen the picture.

In conclusion, we hypothesize that in this patient severe exertion could have caused a mechanical stress on RCA, between the aortic root and pulmonary trunk, leading to myocardial ischemia. Supply-demand imbalance should be kept in mind as a possible cause of chest pain in such individuals.

Disclosures
Informed Consent: Written informed consent was obtained from the patient who participated in this study.
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Conflict of Interest: The authors declare that there is no conflict of interest.


References